Walnut extract inhibits the fibrillization of amyloid beta-protein, and also defibrillizes its preformed fibrils.

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Abstract
Fibrillar amyloid beta-protein (Abeta) is the principal component of amyloid plaques in the brains of patients with Alzheimer's disease. We have studied the effect of walnut extract on Abeta fibrillization by Thioflavin T fluorescence spectroscopy and electron microscopy. The walnut extract not only inhibited Abeta fibril formation in a concentration and time-dependent manner but it was also able to defibrillize Abeta preformed fibrils. Over 90% inhibition of Abeta fibrillization was observed with 5 microl of methanolic extract of walnut (MEOW) both after 2 and 3 days of incubation. The maximum defibrillization (91.6%) was observed when preformed Abeta fibrils were incubated with 10 microl of MEOW for 2 days. These results suggest that walnuts may reduce the risk or delay the onset of Alzheimer's disease by maintaining Abeta in the soluble form. Further studies showed that anti-amyloidogenic compound in walnut is an organic compound of molecular weight less than 10 kDa, which is neither a lipid nor a protein. Chloroform extract of walnut had no effect on Abeta fibrillization while MEOW and its 10 kDa filtrate inhibited Abeta fibrillization equally. It is proposed that polyphenolic compounds (such as flavonoids) present in walnuts may be responsible for its anti-amyloidogenic activity.

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